



844AR Acrylic ESD Coating (Aerosol)

Mektronics

Version No: 2.4

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: 26/01/2022

Print Date: 26/01/2022

L.GHS.AUS.EN

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	844AR
Synonyms	SDS Code: 844AR-Aerosol; 844AR-340G ; UFI:AKM0-900P-200W-7YA4
Other means of identification	Acrylic ESD Coating (Aerosol)

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Static protection for electronic components
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Details of the supplier of the safety data sheet

Registered company name	Mektronics	MG Chemicals (Head office)
Address	Unit 3 8 Bonz Place, Seven Hills NSW 2147 Australia	9347 - 193 Street Surrey V4N 4E7 British Columbia Canada
Telephone	1300 788 701	+(1) 800-201-8822
Fax	1300 722 004	+(1) 800-708-9888
Website	www.mektronics.com.au	www.mgchemicals.com
Email	sales@mektronics.com.au	Info@mgchemicals.com

Emergency telephone number

Association / Organisation	Verisk 3E (Access Code: 335388)
Emergency telephone numbers	+61 1 800 686 951
Other emergency telephone numbers	+61 280363166

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3, Aerosols Category 2, Serious Eye Damage/Eye Irritation Category 1, Carcinogenicity Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
Signal word	Danger

Hazard statement(s)

AUH066	Repeated exposure may cause skin dryness and cracking.
H336	May cause drowsiness or dizziness.
H223+H229	Flammable aerosol; Pressurized container: may burst if heated.
H318	Causes serious eye damage.
H351	Suspected of causing cancer.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.

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P211	Do not spray on an open flame or other ignition source.
P251	Do not pierce or burn, even after use.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P261	Avoid breathing gas

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 Composition / information on ingredients**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
67-64-1	37	<u>acetone</u>
616-38-6	22	<u>dimethyl carbonate</u>
74-98-6	13	<u>propane</u>
75-28-5.	7	<u>iso-butane</u>
108-65-6	4	<u>propylene glycol monomethyl ether acetate, alpha-isomer</u>
71-36-3	3	<u>n-butanol</u>
13463-67-7	2	<u>titanium dioxide</u>

Legend: 1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available

SECTION 4 First aid measures**Description of first aid measures**

Eye Contact	If aerosols come in contact with the eyes: ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If solids or aerosol mists are deposited upon the skin: ▶ Flush skin and hair with running water (and soap if available). ▶ Remove any adhering solids with industrial skin cleansing cream. ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.
Inhalation	If aerosols, fumes or combustion products are inhaled: ▶ Remove to fresh air. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

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For acute or short term repeated exposures to acetone:

- ▶ Symptoms of acetone exposure approximate ethanol intoxication.
- ▶ About 20% is expired by the lungs and the rest is metabolised. Alveolar air half-life is about 4 hours following two hour inhalation at levels near the Exposure Standard; in overdose, saturable metabolism and limited clearance, prolong the elimination half-life to 25-30 hours.
- ▶ There are no known antidotes and treatment should involve the usual methods of decontamination followed by supportive care.

[Ellenhorn and Barceloux: Medical Toxicology]

Management:

Measurement of serum and urine acetone concentrations may be useful to monitor the severity of ingestion or inhalation.

Inhalation Management:

- ▶ Maintain a clear airway, give humidified oxygen and ventilate if necessary.
- ▶ If respiratory irritation occurs, assess respiratory function and, if necessary, perform chest X-rays to check for chemical pneumonitis.
- ▶ Consider the use of steroids to reduce the inflammatory response.
- ▶ Treat pulmonary oedema with PEEP or CPAP ventilation.

Dermal Management:

- ▶ Remove any remaining contaminated clothing, place in double sealed, clear bags, label and store in secure area away from patients and staff.
- ▶ Irrigate with copious amounts of water.
- ▶ An emollient may be required.

Eye Management:

- ▶ Irrigate thoroughly with running water or saline for 15 minutes.
- ▶ Stain with fluorescein and refer to an ophthalmologist if there is any uptake of the stain.

Oral Management:

- ▶ No GASTRIC LAVAGE OR EMETIC

- ▶ Encourage oral fluids.

Systemic Management:

- ▶ Monitor blood glucose and arterial pH.
- ▶ Ventilate if respiratory depression occurs.
- ▶ If patient unconscious, monitor renal function.
- ▶ Symptomatic and supportive care.

The Chemical Incident Management Handbook:

Guy's and St. Thomas' Hospital Trust, 2000

BIOLOGICAL EXPOSURE INDEX

These represent the determinants observed in specimens collected from a healthy worker exposed at the Exposure Standard (ES or TLV):

Determinant	Sampling Time	Index	Comments
Acetone in urine	End of shift	50 mg/L	NS

NS: Non-specific determinant; also observed after exposure to other material

SECTION 5 Firefighting measures

Extinguishing media

- ▶ Alcohol stable foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.
- ▶ Water spray or fog - Large fires only.

SMALL FIRE:

- ▶ Water spray, dry chemical or CO₂

LARGE FIRE:

- ▶ Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	
	carbon dioxide (CO ₂) metal oxides other pyrolysis products typical of burning organic material.
Fire/Explosion Hazard	Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions. BEWARE: Empty solvent, paint, lacquer and flammable liquid drums present a severe explosion hazard if cut by flame torch or welded. Even when thoroughly cleaned or reconditioned the drum seams may retain sufficient solvent to generate an explosive atmosphere in the drum. WARNING: Aerosol containers may present pressure related hazards.

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up. ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely.
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Major Spills	<ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse / absorb vapour. ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. ▶ Collect residues and seal in labelled drums for disposal.
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Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ For low viscosity materials (i) : Drums and jerry cans must be of the non-removable head type. (ii) : Where a can is to be used as an inner package, the can must have a screwed enclosure. ▶ For materials with a viscosity of at least 2680 cSt. (23 deg. C) ▶ For manufactured product having a viscosity of at least 250 cSt. (23 deg. C) ▶ Manufactured product that requires stirring before use and having a viscosity of at least 20 cSt (25 deg. C): (i) Removable head packaging; (ii) Cans with friction closures and (iii) low pressure tubes and cartridges may be used. ▶ Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages ▶ In addition, where inner packagings are glass and contain liquids of packing group I there must be sufficient inert absorbent to absorb any spillage, unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic. ▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Carbonates are incompatible with cerium compounds, germanium, lead diacetate, magnesium, mercurous chloride, silver nitrate ▶ Acetone: <ul style="list-style-type: none"> ▶ may react violently with chloroform, activated charcoal, aliphatic amines, bromine, bromine trifluoride, chlorotriazine, chromic(IV) acid, chromic(VI) acid, chromium trioxide, chromyl chloride, hexachloromelamine, iodine heptafluoride, iodoform, liquid oxygen, nitrosyl chloride, nitrosyl perchlorate, nitryl perchlorate, perchloromelamine, peroxomonosulfuric acid, platinum, potassium tert-butoxide, strong acids, sulfur dichloride, trichloromelamine, xenon tetrafluoride ▶ reacts violently with bromoform and chloroform in the presence of alkalies or in contact with alkaline surfaces. ▶ may form unstable and explosive peroxides in contact with strong oxidisers, fluorine, hydrogen peroxide (90%), sodium perchlorate, 2-methyl-1,3-butadiene ▶ can increase the explosive sensitivity of nitromethane on contact flow or agitation may generate electrostatic charges due to low conductivity ▶ dissolves or attacks most rubber, resins, and plastics (polyethylenes, polyester, vinyl ester, PVC, Neoprene, Viton) ▶ Butane/ isobutane <ul style="list-style-type: none"> ▶ reacts violently with strong oxidisers ▶ reacts with acetylene, halogens and nitrous oxides ▶ is incompatible with chlorine dioxide, conc. nitric acid and some plastics ▶ may generate electrostatic charges, due to low conductivity, in flow or when agitated - these may ignite the vapour. ▶ Segregate from nickel carbonyl in the presence of oxygen, heat (20-40 C) <ul style="list-style-type: none"> ▶ Esters react with acids to liberate heat along with alcohols and acids. ▶ Strong oxidising acids may cause a vigorous reaction with esters that is sufficiently exothermic to ignite the reaction products. ▶ Heat is also generated by the interaction of esters with caustic solutions. ▶ Flammable hydrogen is generated by mixing esters with alkali metals and hydrides. ▶ Esters may be incompatible with aliphatic amines and nitrates. ▶ Ketones in this group: <ul style="list-style-type: none"> ▶ are reactive with many acids and bases liberating heat and flammable gases (e.g., H₂). ▶ react with reducing agents such as hydrides, alkali metals, and nitrides to produce flammable gas (H₂) and heat. ▶ are incompatible with isocyanates, aldehydes, cyanides, peroxides, and anhydrides. ▶ react violently with aldehydes, HNO₃ (nitric acid), HNO₃ + H₂O₂ (mixture of nitric acid and hydrogen peroxide), and HClO₄ (perchloric acid). ▶ may react with hydrogen peroxide to form unstable peroxides; many are heat- and shock-sensitive explosives. <p>A significant property of most ketones is that the hydrogen atoms on the carbons next to the carbonyl group are relatively acidic when compared to hydrogen atoms in typical hydrocarbons. Under strongly basic conditions these hydrogen atoms may be abstracted to form an enolate anion. This property allows ketones, especially methyl ketones, to participate in condensation reactions with other ketones and aldehydes. This type of</p>

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condensation reaction is favoured by high substrate concentrations and high pH (greater than 1 wt% NaOH).
 Propane:
 ▶ reacts violently with strong oxidisers, barium peroxide, chlorine dioxide, dichlorine oxide, fluorine etc.
 ▶ liquid attacks some plastics, rubber and coatings
 ▶ may accumulate static charges which may ignite its vapours
 ▶ Segregate from alcohol, water.
 ▶ Avoid reaction with oxidising agents, bases and strong reducing agents.

SECTION 8 Exposure controls / personal protection**Control parameters****Occupational Exposure Limits (OEL)****INGREDIENT DATA**

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	acetone	Acetone	500 ppm / 1185 mg/m3	2375 mg/m3 / 1000 ppm	Not Available	Not Available
Australia Exposure Standards	propylene glycol monomethyl ether acetate, alpha-isomer	1-Methoxy-2-propanol acetate	50 ppm / 274 mg/m3	548 mg/m3 / 100 ppm	Not Available	Not Available
Australia Exposure Standards	n-butanol	n-Butyl alcohol	Not Available	Not Available	50 ppm / 152 mg/m3	Not Available
Australia Exposure Standards	titanium dioxide	Titanium dioxide	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
acetone	Not Available	Not Available	Not Available
dimethyl carbonate	11 ppm	120 ppm	700 ppm
propane	Not Available	Not Available	Not Available
iso-butane	5500* ppm	17000** ppm	53000*** ppm
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available	Not Available
n-butanol	60 ppm	800 ppm	8000** ppm
titanium dioxide	30 mg/m3	330 mg/m3	2,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
acetone	2,500 ppm	Not Available
dimethyl carbonate	Not Available	Not Available
propane	2,100 ppm	Not Available
iso-butane	Not Available	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available	Not Available
n-butanol	1,400 ppm	Not Available
titanium dioxide	5,000 mg/m3	Not Available

MATERIAL DATA

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class OSF Description

A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities

B 26-550 As 'A' for 50-90% of persons being distracted

C 1-26 As 'A' for less than 50% of persons being distracted

D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached

E <0.18 As 'D' for less than 10% of persons aware of being tested

Odour Threshold Value: 3.6 ppm (detection), 699 ppm (recognition)

Saturation vapour concentration: 237000 ppm @ 20 C

NOTE: Detector tubes measuring in excess of 40 ppm, are available.

Exposure at or below the recommended TLV-TWA is thought to protect the worker against mild irritation associated with brief exposures and the bioaccumulation, chronic irritation of the respiratory tract and headaches associated with long-term acetone exposures. The NIOSH REL-TWA is substantially lower and has taken into account slight irritation experienced by volunteer subjects at 300 ppm. Mild irritation to acclimatised workers begins at about 750 ppm - unacclimatised subjects will experience irritation at about 350-500 ppm but acclimatisation can occur rapidly. Disagreement between the peak bodies is based largely on the view by ACGIH that widespread use of acetone, without evidence of significant adverse health effects at higher concentrations, allows acceptance of a higher limit.

Half-life of acetone in blood is 3 hours which means that no adjustment for shift-length has to be made with reference to the standard 8 hour/day, 40 hours per week because body clearance occurs within any shift with low potential for accumulation.

A STEL has been established to prevent excursions of acetone vapours that could cause depression of the central nervous system.

Odour Safety Factor(OSF)

Continued...

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OSF=38 (ACETONE)

Animals exposed by inhalation to 10 mg/m³ titanium dioxide show no significant fibrosis, possibly reversible tissue reaction. The architecture of lung air spaces remains intact.

. The label on a package containing 1% or more of titanium oxide with aerodynamic diameter equal or below 10 microns shall bear the following statement: EUH211 'Warning! Hazardous respirable droplets may be formed when sprayed. Do NOT breathe spray or mist'

. The label on the packaging of solid mixtures containing 1% or more of titanium dioxide shall bear the following statement: EUH212 'Warning! Hazardous respirable dust may be formed when used. Do not breathe dust'.

In addition, the label on the packaging of liquid and solid mixtures not intended for the general public and not classified as hazardous which are labelled EUH211 or EU212 shall bear statement EUH210: 'Safety data sheet available on request.'

For butane:

Odour Threshold Value: 2591 ppm (recognition)

Butane in common with other homologues in the straight chain saturated aliphatic hydrocarbon series is not characterised by its toxicity but by its narcosis-inducing effects at high concentrations. The TLV is based on analogy with pentane by comparing their lower explosive limits in air. It is concluded that this limit will protect workers against the significant risk of drowsiness and other narcotic effects.

Odour Safety Factor(OSF)

OSF=0.22 (n-BUTANE)

for propylene glycol monomethyl ether acetate (PGMEA)

Saturated vapour concentration: 4868 ppm at 20 C.

A two-week inhalation study found nasal effects to the nasal mucosa in animals at concentrations up to 3000 ppm. Differences in the teratogenic potential of the alpha (commercial grade) and beta isomers of PGMEA may be explained by the formation of different metabolites. The beta-isomer is thought to be oxidised to methoxypropionic acid, a homologue to methoxycetic acid which is a known teratogen. The alpha- form is conjugated and excreted. PGMEA mixture (containing 2% to 5% beta isomer) is a mild skin and eye irritant, produces mild central nervous system effects in animals at 3000 ppm and produces mild CNS impairment and upper respiratory tract and eye irritation in humans at 1000 ppm. In rats exposed to 3000 ppm PGMEA produced slight foetotoxic effects (delayed sternabral ossification) - no effects on foetal development were seen in rabbits exposed at 3000 ppm.

For propane

Odour Safety Factor(OSF)

OSF=0.16 (PROPANE)

For n-butanol:

Odour Threshold Value: 0.12-3.4 ppm (detection), 1.0-3.5 ppm (recognition)

NOTE: Detector tubes for n-butanol, measuring in excess of 5 ppm are commercially available.

Exposure at or below the TLV-TWA is thought to provide protection against hearing loss due to vestibular and auditory nerve damage in younger workers and to protect against the significant risk of headache and irritation.

25 ppm may produce mild irritation of the respiratory tract 50 ppm may produce headache and vertigo.

Higher concentrations may produce marked irritation, sore throat, coughing, nausea, shortness of breath, pulmonary injury and central nervous system depression characterised by headache, dizziness, dullness and drowsiness.

6000 ppm may produce giddiness, prostration, narcosis, ataxia, and death.

Odour Safety Factor (OSF)

OSF=60 (n-BUTANOL)

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard 'physically' away from the worker and ventilation that strategically 'adds' and 'removes' air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection.</p> <p>Provide adequate ventilation in warehouse or closed storage areas.</p> <p>Air contaminants generated in the workplace possess varying 'escape' velocities which, in turn, determine the 'capture velocities' of fresh circulating air required to effectively remove the contaminant.</p>										
	Type of Contaminant:	Speed:									
	aerosols, (released at low velocity into zone of active generation)	0.5-1 m/s									
	direct spray, spray painting in shallow booths, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)									
	Within each range the appropriate value depends on:										
	<table border="1"> <thead> <tr> <th>Lower end of the range</th><th>Upper end of the range</th></tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td><td>1: Disturbing room air currents</td></tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td><td>2: Contaminants of high toxicity</td></tr> <tr> <td>3: Intermittent, low production.</td><td>3: High production, heavy use</td></tr> <tr> <td>4: Large hood or large air mass in motion</td><td>4: Small hood-local control only</td></tr> </tbody> </table>		Lower end of the range	Upper end of the range	1: Room air currents minimal or favourable to capture	1: Disturbing room air currents	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity	3: Intermittent, low production.	3: High production, heavy use	4: Large hood or large air mass in motion
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<p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>											
   											

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Eye and face protection	<p>No special equipment for minor exposure i.e. when handling small quantities.</p> <p>OTHERWISE: For potentially moderate or heavy exposures:</p> <ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ NOTE: Contact lenses pose a special hazard; soft lenses may absorb irritants and ALL lenses concentrate them. ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<p>Wear general protective gloves, eg. light weight rubber gloves.</p> <p>For esters:</p> <ul style="list-style-type: none"> ▶ Do NOT use natural rubber, butyl rubber, EPDM or polystyrene-containing materials. ▶ No special equipment needed when handling small quantities. ▶ OTHERWISE: ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear. ▶ Insulated gloves: <p>NOTE: Insulated gloves should be loose fitting so that may be removed quickly if liquid is spilled upon them. Insulated gloves are not made to permit hands to be placed in the liquid; they provide only short-term protection from accidental contact with the liquid.</p>
Body protection	See Other protection below
Other protection	<p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces.

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

Forsberg Clothing Performance Index.

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
PE/EVAL/PE	A
TEFLON	B
BUTYL	C
BUTYL/NEOPRENE	C
CPE	C
HYPALON	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
NITRILE+PVC	C
PE	C
PVA	C
PVC	C
PVDC/PE/PVDC	C
SARANEX-23	C
SARANEX-23 2-PLY	C
VITON/NEOPRENE	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as 'feel' or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

Type AX Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the 'Exposure Standard' (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AX-AUS	-	AX-PAPR-AUS / Class 1
up to 50 x ES	-	AX-AUS / Class 1	-
up to 100 x ES	-	AX-2	AX-PAPR-2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used
- ▶ Generally not applicable.

Aerosols, in common with most vapours/ mists, should never be used in confined spaces without adequate ventilation. Aerosols, containing agents designed to enhance or mask smell, have triggered allergic reactions in predisposed individuals.

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	AX-AUS / Class 1	-

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844AR Acrylic ESD Coating (Aerosol)

up to 50	1000	-	AX-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	AX-2
up to 100	10000	-	AX-3
100+		-	Airline**

** - Continuous-flow or positive pressure demand.

A(All classes) = Organic vapours, B AUS or B1 = Acid gases, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO₂), G = Agricultural chemicals, K = Ammonia(NH₃), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 deg C)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Off white		
Physical state	Liquified Gas	Relative density (Water = 1)	0.95
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	345
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	<20.5
Initial boiling point and boiling range (°C)	>56	Molecular weight (g/mol)	Not Available
Flash point (°C)	-18	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	9.5	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	2.1	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	350	Gas group	Not Available
Solubility in water	Partly miscible	pH as a solution (Not Available%)	Not Available
Vapour density (Air = 1)	>1.5	VOC g/L	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	► Elevated temperatures. ► Presence of open flame. ► Product is considered stable. ► Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
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844AR Acrylic ESD Coating (Aerosol)

	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>The main effects of simple aliphatic esters are narcosis and irritation and anaesthesia at higher concentrations. These effects become greater as the molecular weights and boiling points increase. Central nervous system depression, headache, drowsiness, dizziness, coma and neurobehavioral changes may also be symptomatic of overexposure. Respiratory tract involvement may produce mucous membrane irritation, dyspnea, and tachypnoea, pharyngitis, bronchitis, pneumonitis and, in massive exposures, pulmonary oedema (which may be delayed). Gastrointestinal effects include nausea, vomiting, diarrhoea and abdominal cramps. Liver and kidney damage may result from massive exposures.</p> <p>No health effects were seen in humans exposed at 1,000 ppm isobutane for up to 8 hours or 500 ppm for 8 hours/day for 10 days. Isobutane can have anaesthetic and asphyxiant effects at high concentrations, well above the lower explosion limit of 1.8% (18,000 ppm).</p> <p>Butane is a simple asphyxiant and is mildly anaesthetic at high concentrations (20-25%). 10000 ppm for 10 minutes causes drowsiness. Narcotic effects may be accompanied by exhilaration, dizziness, headache, nausea, confusion, incoordination and unconsciousness in severe cases.</p> <p>The paraffin gases C1-4 are practically nontoxic below the lower flammability limit, 18,000 to 50,000 ppm; above this, low to moderate incidental effects such as CNS depression and irritation occur, but are completely reversible upon cessation of the exposure.</p> <p>The vapour is discomforting</p> <p>WARNING:Intentional misuse by concentrating/inhaling contents may be lethal.</p> <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.</p> <p>The use of a quantity of material in an unventilated or confined space may result in increased exposure and an irritating atmosphere developing. Before starting consider control of exposure by mechanical ventilation.</p> <p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Exposure to ketone vapours may produce nose, throat and mucous membrane irritation. High concentrations of vapour may produce central nervous system depression characterised by headache, vertigo, loss of coordination, narcosis and cardiorespiratory failure. Some ketones produce neurological disorders (polyneuropathy) characterised by bilateral symmetrical paresthesia and muscle weakness primarily in the legs and arms.</p> <p>Systemic effects of acetone inhalation exposure include central nervous system depression, light-headedness, incoherent speech, ataxia, stupor, hypotension, tachycardia, metabolic acidosis, hyperglycaemia and ketosis. Rarely, convulsions and tubular necrosis may be evident. Other symptoms of exposure may include restlessness, headache, vomiting, low blood-pressure and rapid and irregular pulse, eye and throat irritation, weakness of the legs and dizziness. Inhalation of high concentrations may produce dryness of the mouth and throat, nausea, uncoordinated movement, loss of coordinated speech, drowsiness and, in severe cases, coma. Inhalation of acetone vapours over long periods causes irritation of the respiratory tract, coughing and headache. Rats exposed to 52200 ppm vapour for 1 hour showed clear signs of narcosis; fatalities occurred at 126600 ppm.</p>
Ingestion	<p>The material has NOT been classified by EC Directives or other classification systems as 'harmful by ingestion'. This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Many aliphatic hydrocarbons create a burning sensation because they are irritating to the GI mucosa. Vomiting has been reported in up to one third of all hydrocarbon exposures. While most aliphatic hydrocarbons have little GI absorption, aspiration frequently occurs, either initially or in a semi-delayed fashion as the patient coughs or vomits, thereby resulting in pulmonary effects. Once aspirated, the hydrocarbons can create a severe pneumonitis.</p> <p>Rats given isoparaffinic hydrocarbons - isoalkanes- (after 18-24 hours fasting) showed lethargy and/or general weakness, ataxia and diarrhoea. Symptoms disappeared within 24-28 hours.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
Skin Contact	<p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Spray mist may produce discomfort</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects.</p> <p>Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p> <p>The material may produce mild skin irritation; limited evidence or practical experience suggests, that the material either:</p> <ul style="list-style-type: none"> ▶ produces mild inflammation of the skin in a substantial number of individuals following direct contact, and/or ▶ produces significant, but mild, inflammation when applied to the healthy intact skin of animals (for up to four hours), such inflammation being present twenty-four hours or more after the end of the exposure period. <p>Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (non allergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p>
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	<p>Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>There is sufficient evidence to establish a causal relationship between human exposure to the material and impaired fertility.</p> <p>Prolonged or repeated skin contact may cause drying with cracking, irritation and possible dermatitis following.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p>

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	Workers exposed to 700 ppm acetone for 3 hours/day for 7-15 years showed inflammation of the respiratory tract, stomach and duodenum, attacks of giddiness and loss of strength. Exposure to acetone may enhance liver toxicity of chlorinated solvents.	
844AR Acrylic ESD Coating (Aerosol)	TOXICITY Not Available	IRRITATION Not Available
acetone	TOXICITY Dermal (rabbit) LD50: 20000 mg/kg ^[2] Inhalation(Mouse) LC50; 44 mg/L4h ^[2] Oral (Rat) LD50; 5800 mg/kg ^[2]	IRRITATION Eye (human): 500 ppm - irritant Eye (rabbit): 20mg/24hr -moderate Eye (rabbit): 3.95 mg - SEVERE Eye: adverse effect observed (irritating) ^[1] Skin (rabbit): 500 mg/24hr - mild Skin (rabbit):395mg (open) - mild Skin: no adverse effect observed (not irritating) ^[1]
dimethyl carbonate	TOXICITY Dermal (rabbit) LD50: >2000 mg/kg ^[1] Inhalation(Rat) LC50; >5.36 mg/l4h ^[1] Oral (Rat) LD50; >5000 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
propane	TOXICITY Inhalation(Rat) LC50; >13023 ppm4h ^[1]	IRRITATION Not Available
iso-butane	TOXICITY Inhalation(Rat) LC50; >13023 ppm4h ^[1]	IRRITATION Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	TOXICITY dermal (rat) LD50: >2000 mg/kg ^[1] Oral (Rat) LD50; 3739 mg/kg ^[2]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
n-butanol	TOXICITY Dermal (rabbit) LD50: 3400 mg/kg ^[2] Inhalation(Rat) LC50; 8000 ppm4h ^[2] Oral (Rat) LD50; 790 mg/kg ^[2]	IRRITATION Eye (human): 50 ppm - irritant Eye (rabbit): 1.6 mg-SEVERE Eye (rabbit): 24 mg/24h-SEVERE Eye: adverse effect observed (irreversible damage) ^[1] Skin (rabbit): 405 mg/24h-moderate Skin: adverse effect observed (irritating) ^[1]
titanium dioxide	TOXICITY dermal (hamster) LD50: >=10000 mg/kg ^[2] Inhalation(Rat) LC50; >2.28 mg/l4h ^[1] Oral (Rat) LD50; >=2000 mg/kg ^[1]	IRRITATION Eye: no adverse effect observed (not irritating) ^[1] Skin (human): 0.3 mg /3D (int)-mild * Skin: no adverse effect observed (not irritating) ^[1]
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances	
844AR Acrylic ESD Coating (Aerosol)	<p>Generally, linear and branched-chain alkyl esters are hydrolysed to their component alcohols and carboxylic acids in the intestinal tract, blood and most tissues throughout the body. Following hydrolysis the component alcohols and carboxylic acids are metabolized. Oral acute toxicity studies have been reported for 51 of the 67 esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids. The very low oral acute toxicity of this group of esters is demonstrated by oral LD50 values greater than 1850 mg/kg bw. Genotoxicity studies have been performed in vitro using the following esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids: methyl acetate, butyl acetate, butyl stearate and the structurally related isoamyl formate and demonstrates that these substances are not genotoxic.</p> <p>The JECFA Committee concluded that the substances in this group would not present safety concerns at the current levels of intake the esters of aliphatic acyclic primary alcohols and aliphatic linear saturated carboxylic acids are generally used as flavouring substances up to average maximum levels of 200 mg/kg. Higher levels of use (up to 3000 mg/kg) are permitted in food categories such as chewing gum and hard candy. In Europe the upper use levels for these flavouring substances are generally 1 to 30 mg/kg foods and in special food categories like candy and alcoholic beverages up to 300 mg/kg foods</p> <p>International Program on Chemical Safety: the Joint FAO/WHO Expert Committee on Food Additives (JECFA)</p>	

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	Esters of Aliphatic acyclic primary alcohols with aliphatic linear saturated carboxylic acids.; 1998
	<p>A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects. The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I] *Shin-Etsu SDS for propylene glycol ethers (PGEs):</p> <p>Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM).</p> <p>Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids.</p> <p>Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects).</p> <p>This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product.</p> <p>Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body.</p> <p>As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces.</p> <p>As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM).</p> <p>Inhalation LC50 values were higher than 5,000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the remaining category members are slightly to non-irritating. None are skin sensitizers.</p> <p>In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).</p> <p>Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.</p> <p>One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. in a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.</p> <p>In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.</p> <p>The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. <i>In vitro</i>, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic <i>in vivo</i>. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice. A BASF report (in ECETOC) showed that inhalation exposure to 545 ppm PGMEA (beta isomer) was associated with a teratogenic response in rabbits; but exposure to 145 ppm and 36 ppm had no adverse effects.</p> <p>The beta isomer of PGMEA comprises only 10% of the commercial material, the remaining 90% is alpha isomer. Hazard appears low but emphasizes the need for care in handling this chemical. [I.C.I]</p>
	<p>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.</p> <p>for n-butanol</p> <p>Acute toxicity: n-Butanol (BA) was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg. Different strains of rat were used in each of four studies, which may account for the variability. Oral LD50 values for mice, rabbits, hamsters, dogs, and male rats all fell within the same range. The rat inhalation LC₀ of 8000 ppm (24000 mg/m³) indicates very low inhalation toxicity (no lethality at 8000 ppm). The rabbit dermal LD50 was 3402 mg/kg, indicating that BA can penetrate the skin, but not very readily. Animal experiments and human experience indicate that BA is, at most, moderately irritating to the skin, but it is a severe eye irritant. These effects are most likely due to BA's localised defatting and drying characteristics. Although no animal data are available, human studies and experience show that BA is not likely to be a skin sensitizer.</p> <p>The median odor threshold for BA (0.17 ppm) is well below the lowest nasal irritation threshold in humans (289 ppm), allowing warning of possible chemical exposure prior to nasal irritation occurring. Human studies are complicated by the odor characteristics of the material, as the odor threshold is well below the levels at which irritation is observed.</p> <p>Repeat dose toxicity: An <i>in vivo</i> toxicokinetics study confirmed the rapid metabolism of n-butyl acetate (BAC) to BA. Hydrolysis of BAC in blood and brain was estimated to be 99 percent complete within 2.7 minutes (elimination t_{1/2} = 0.41 minute). Thus, organisms exposed to BAC can experience appreciable tissue concentrations of BA. In this</p>
N-BUTANOL	

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	<p>way, the results of toxicity studies with BAc can be used as supplemental, surrogate data to provide information on the toxicity of BA.</p> <p>A thirteen-week, subchronic exposure to BAc, the metabolic precursor of BA, produced transient hypoactivity (during exposure only) at 1500 and 3000 ppm (7185 and 14370 mg/m³) along with decreased body weight and food consumption, but no post exposure neurotoxicity even at 3000 ppm. A concurrent subchronic neurotoxicity study under the same exposure conditions showed no evidence of cumulative neurotoxicity based upon functional observational battery endpoints, quantitative motor activity, neuropathology and scheduled-controlled operant behavior endpoints. A no observable effect level (NOAEL) of 500 ppm (2395 mg/m³) was reported for systemic effects in rats, and a NOAEL of 3000 ppm (14370 mg/m³) was reported for post exposure neurotoxicity in rats.</p> <p>Reproductive toxicity: Several studies indicate that BA is not a reproductive toxicant.</p> <p>Female rats exposed to 6000 ppm (18000 mg/m³) BA throughout gestation and male rats exposed to 6000 ppm (18000 mg/m³) BA for six weeks prior to mating showed no effects on fertility or pregnancy rate. Male rats given BA at 533 mg/kg/day for 5 days had no testicular toxicity.</p> <p>Developmental toxicity: BA produced only mild foetotoxicity and developmental alterations at or near the maternally toxic (even lethal) dose of 8000 ppm (24000 mg/m³) throughout gestation.</p> <p>Genotoxicity: An entire battery of negative in vitro tests and a negative in vivo micronucleus test indicate that BA is not genotoxic.</p> <p>Carcinogenicity: Based upon the battery of negative mutagenicity and clastogenicity findings, BA presents a very small potential for carcinogenicity.</p>
TITANIUM DIOXIDE	<p>* IUCLID</p> <p>Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.</p> <p>The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.</p> <p>WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.</p>
844AR Acrylic ESD Coating (Aerosol) & N-BUTANOL & TITANIUM DIOXIDE	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
844AR Acrylic ESD Coating (Aerosol) & TITANIUM DIOXIDE	<p>For titanium dioxide:</p> <p>Humans can be exposed to titanium dioxide via inhalation, ingestion or dermal contact. In human lungs, the clearance kinetics of titanium dioxide is poorly characterized relative to that in experimental animals. (General particle characteristics and host factors that are considered to affect deposition and retention patterns of inhaled, poorly soluble particles such as titanium dioxide are summarized in the monograph on carbon black.) With regard to inhaled titanium dioxide, human data are mainly available from case reports that showed deposits of titanium dioxide in lung tissue as well as in lymph nodes. A single clinical study of oral ingestion of fine titanium dioxide showed particle size-dependent absorption by the gastrointestinal tract and large interindividual variations in blood levels of titanium dioxide. Studies on the application of sunscreens containing ultrafine titanium dioxide to healthy skin of human volunteers revealed that titanium dioxide particles only penetrate into the outermost layers of the stratum corneum, suggesting that healthy skin is an effective barrier to titanium dioxide. There are no studies on penetration of titanium dioxide in compromised skin.</p> <p>Respiratory effects that have been observed among groups of titanium dioxide-exposed workers include decline in lung function, pleural disease with plaques and pleural thickening, and mild fibrotic changes. However, the workers in these studies were also exposed to asbestos and/or silica.</p> <p>No data were available on genotoxic effects in titanium dioxide-exposed humans.</p> <p>Many data on deposition, retention and clearance of titanium dioxide in experimental animals are available for the inhalation route. Titanium dioxide inhalation studies showed differences — both for normalized pulmonary burden (deposited mass per dry lung, mass per body weight) and clearance kinetics — among rodent species including rats of different size, age and strain. Clearance of titanium dioxide is also affected by pre-exposure to gaseous pollutants or co-exposure to cytotoxic aerosols. Differences in dose rate or clearance kinetics and the appearance of focal areas of high particle burden have been implicated in the higher toxic and inflammatory lung responses to intratracheally instilled vs inhaled titanium dioxide particles. Experimental studies with titanium dioxide have demonstrated that rodents experience dose-dependent impairment of alveolar macrophage-mediated clearance. Hamsters have the most efficient clearance of inhaled titanium dioxide. Ultrafine primary particles of titanium dioxide are more slowly cleared than their fine counterparts.</p> <p>Titanium dioxide causes varying degrees of inflammation and associated pulmonary effects including lung epithelial cell injury, cholesterol granulomas and fibrosis. Rodents experience stronger pulmonary effects after exposure to ultrafine titanium dioxide particles compared with fine particles on a mass basis. These differences are related to lung burden in terms of particle surface area, and are considered to result from impaired phagocytosis and sequestration of ultrafine particles into the interstitium.</p> <p>Fine titanium dioxide particles show minimal cytotoxicity to and inflammatory/pro-fibrotic mediator release from primary human alveolar macrophages in vitro compared with other particles. Ultrafine titanium dioxide particles inhibit phagocytosis of alveolar macrophages in vitro at mass dose concentrations at which this effect does not occur with fine titanium dioxide. In-vitro studies with fine and ultrafine titanium dioxide and purified DNA show induction of DNA damage that is suggestive of the generation of reactive oxygen species by both particle types. This effect is stronger for ultrafine than for fine titanium dioxide, and is markedly enhanced by exposure to simulated sunlight/ultraviolet light.</p> <p>Animal carcinogenicity data</p> <p>Pigmentary and ultrafine titanium dioxide were tested for carcinogenicity by oral administration in mice and rats, by inhalation in rats and female mice, by intratracheal administration in hamsters and female rats and mice, by subcutaneous injection in rats and by intraperitoneal administration in male mice and female rats.</p> <p>In one inhalation study, the incidence of benign and malignant lung tumours was increased in female rats. In another inhalation study, the incidences of lung adenomas were increased in the high-dose groups of male and female rats. Cystic keratinizing lesions that were diagnosed as squamous-cell carcinomas but re-evaluated as non-neoplastic pulmonary keratinizing cysts were also observed in the high-dose groups of female rats. Two inhalation studies in rats and one in female mice were negative.</p> <p>Intratracheally instilled female rats showed an increased incidence of both benign and malignant lung tumours following treatment with two types of titanium dioxide. Tumour incidence was not increased in intratracheally instilled hamsters and female mice.</p> <p>In-vivo studies have shown enhanced micronucleus formation in bone marrow and peripheral blood lymphocytes of intraperitoneally instilled mice. Increased Hprt mutations were seen in lung epithelial cells isolated from titanium dioxide-instilled rats. In another study, no enhanced oxidative DNA damage was observed in lung tissues of rats that were intratracheally instilled with titanium dioxide. The results of most in-vitro genotoxicity studies with titanium dioxide were negative.</p>
844AR Acrylic ESD Coating (Aerosol) & ACETONE	<p>for acetone:</p> <p>The acute toxicity of acetone is low. Acetone is not a skin irritant or sensitiser but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic</p>

844AR Acrylic ESD Coating (Aerosol)

effects and the effects may have been associated with microsomal enzyme induction. Haematologic effects consistent with macrocytic anaemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m³ and in rats at 26,100 mg/m³. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m³ for both rats and mice.

Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m³, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.

The scientific literature contains many different studies that have measured either the neurobehavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m³ have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m³ were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m³ or greater.

ACETONE & TITANIUM DIOXIDE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.			
PROPANE & TITANIUM DIOXIDE	No significant acute toxicological data identified in literature search.			
Acute Toxicity	✗	Carcinogenicity	✓	
Skin Irritation/Corrosion	✗	Reproductivity	✗	
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓	
Respiratory or Skin sensitisation	✗	STOT - Repeated Exposure	✗	
Mutagenicity	✗	Aspiration Hazard	✗	

Legend: ✗ – Data either not available or does not fill the criteria for classification
✓ – Data available to make classification

SECTION 12 Ecological information**Toxicity**

844AR Acrylic ESD Coating (Aerosol)	Toxicity				
	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
acetone	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	12h	Fish	0.001mg/L	4
	LC50	96h	Fish	3744.6-5000.7mg/L	4
	EC50	48h	Crustacea	6098.4mg/L	5
	EC50	96h	Algae or other aquatic plants	9.873-27.684mg/l	4
dimethyl carbonate	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	25mg/l	2
	LC50	96h	Fish	>=100mg/l	2
	EC50	72h	Algae or other aquatic plants	>57.29mg/l	2
	EC50	48h	Crustacea	>74.16mg/l	2
	EC50	96h	Algae or other aquatic plants	166.6-211mg/l	2
propane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	LC50	96h	Fish	24.11mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2
iso-butane	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	7.71mg/l	2
	LC50	96h	Fish	24.11mg/l	2
	EC50	96h	Algae or other aquatic plants	7.71mg/l	2
propylene glycol monomethyl ether acetate, alpha-isomer	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	336h	Fish	47.5mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	72h	Algae or other aquatic plants	>1000mg/l	2
	EC50	48h	Crustacea	373mg/l	2

Continued...

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	EC50	96h	Algae or other aquatic plants	>1000mg/l	2
n-butanol	Endpoint	Test Duration (hr)	Species	Value	Source
	NOEC(ECx)	504h	Crustacea	4.1mg/l	2
	LC50	96h	Fish	100-500mg/l	4
	EC50	72h	Algae or other aquatic plants	>500mg/l	1
	EC50	48h	Crustacea	>500mg/l	1
	EC50	96h	Algae or other aquatic plants	225mg/l	2
titanium dioxide	Endpoint	Test Duration (hr)	Species	Value	Source
	BCF	1008h	Fish	<1.1-9.6	7
	NOEC(ECx)	504h	Crustacea	0.02mg/l	4
	LC50	96h	Fish	1.85-3.06mg/l	4
	EC50	72h	Algae or other aquatic plants	3.75-7.58mg/l	4
	EC50	48h	Crustacea	1.9mg/l	2
	EC50	96h	Algae or other aquatic plants	179.05mg/l	2

Legend:

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For ketones:

Ketones, unless they are alpha, beta--unsaturated ketones, can be considered as narcosis or baseline toxicity compounds

Hydrolysis may also involve the addition of water to ketones to yield ketals under mild acid conditions. However, this addition of water is thermodynamically favorable only for low molecular weight ketones. This addition is an equilibrium reaction that is reversible upon a change of water concentration and the reaction ultimately leads to no permanent change in the structure of the ketone substrateThe higher molecular weight ketones do not form stable ketals. Therefore, the ketones are stable to water under ambient environmental conditions Another possible reaction of ketones in water involves the enolic hydrogen on the carbons bonded to the carbonyl function. Under conditions of high pH (pH greater than 10), the enolic proton is abstracted by base (OH-) forming a carbanion intermediate that may react with other organic substrates (e.g., ketones, esters, aldehydes) containing a center for nucleophilic attack. The reactions, commonly recognized as condensation reactions, produce higher molecular weight products. Under ambient conditions of temperature, pH, and low concentration, these condensation reactions are unfavorable.

Based on its reactions in air, it seems likely that ketones undergo photolysis in water. It is probable that ketones will be biodegraded to an appreciable degree by micro-organisms in soil and water. They are unlikely to bioconcentrate or biomagnify.

For isobutane:

Refrigerant Gas: Saturated Hydrocarbons have zero ozone depletion potential (ODP) and will photodegrade under atmospheric conditions. [Calor Gas]

Environmental Fate

Terrestrial fate: An estimated Koc value of 35 suggests that isobutane will have very high mobility in soil. Its very high Henry's Law constant, 4.08 atm-cu m/mole, (calculated from its vapor pressure and water solubility, high vapor pressure, 2611 mm Hg at 25 deg C, and low adsorptivity to soil indicate that volatilisation will be an important fate process from both moist and dry soil surfaces. Isobutane is biodegradable, especially under acclimated conditions, and may biodegrade in soil.

Aquatic fate: The estimated Koc value suggests that isobutane would not adsorb to sediment and particulate matter in the water column. Additional evidence that isobutane is not removed to sediment has been obtained from microcosm experiments. Isobutane will readily volatilise from water based on its estimated Henry's Law constant of 4.08 atm-cu m/mole. Estimated half-lives for a model river and model lake are 2.2 hr and 3.0 days, respectively. An estimated BCF value of 74 based on the log Kow suggests that isobutane will not bioconcentrate in aquatic organisms.

Results indicate that gas exchange is the dominant removal mechanism for isobutane gases from the water column following a hypothetical input. The volatilisation half-lives for isobutane from the water columns in natural estuaries are estimated to be 4.4 and 6.8 days at 20 and 10 deg C, respectively.

Isobutane also biodegrades in the microcosm at a rate that is slower than for n-butane and falls between propane and ethane in susceptibility. Biodegradation of isobutane initially occurs with a half-lives of 16-26 days at 20 deg C and 33-139 days at 10 deg C, significantly slower than the loss predicted by gas exchange from typical natural estuaries. However, after a lag of 2-4 weeks, the biodegradation rate increases markedly so that in the case of chronic inputs, biodegradation can become the dominant removal mechanism.

Atmospheric fate: Isobutane is a gas at ordinary temperatures. It is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is 6.9 days, assuming a hydroxyl radical concn of 5x105 radicals per cubic cm. When isobutane was exposed to sunlight for 6 hr in a teflar bag filled with Los Angeles air, 6% of the isobutane degraded. The air contained 4529 ppb-C hydrocarbons and 870 ppb of NOX. The tropospheric loss of volatile hydrocarbons such as isobutane by wet and dry deposition are believed to be of minor importance. Indeed, isobutane assimilated into precipitation may evaporate during transport as well as being reemitted into the atmosphere after deposition. Isobutane is a contributor to the production of PAN (peroxyacetyl nitrates) under photochemical smog conditions

For propane:

Environmental Fate

Terrestrial fate: An estimated Koc value of 460 determined from a log Kow of 2.36 indicates that propane is expected to have moderate mobility in soil. Volatilisation of propane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 7.07x10-1 atm-cu m/mole, derived from its vapor pressure, 7150 mm Hg, and water solubility, 62.4 mg/L. Propane is expected to volatilise from dry soil surfaces based upon its vapor pressure. Using cell suspensions of microorganisms isolated from soil and water, propane was oxidised to acetone within 24 hours, suggesting that biodegradation may be an important fate process in soil and sediment.

Aquatic fate: The estimated Koc value indicates that propane is expected to adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant. Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 41 minutes and 2.6 days, respectively. An estimated BCF of 13.1 using log Kow suggests the potential for bioconcentration in aquatic organisms is low. After 192 hr, the trace concentration of propane contained in gasoline remained unchanged for both a sterile control and a mixed culture sample collected from ground water contaminated with gasoline. This indicates that biodegradation may not be an important fate process in water.

Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and vapour pressure, propane is expected to exist solely as a gas in the ambient atmosphere. Gas-phase propane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 14 days, calculated from its rate constant of 1.15x10-12 cu cm/molecule-sec at 25 deg C. Propane does not contain chromophores that absorb at wavelengths >290 nm and therefore is not expected to be susceptible to direct photolysis by sunlight.

DO NOT discharge into sewer or waterways.

for acetone:

log Kow: -0.24

Half-life (hr) air: 312-1896

Half-life (hr) H2O surface water: 20

Henry's atm m3 /mol: 3.67E-05

BOD 5: 0.31-1.76,46-55%

COD: 1.12-2.07

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ThOD: 2.2

BCF: 0.69

Environmental fate:

Acetone preferentially locates in the air compartment when released to the environment. A substantial amount of acetone can also be found in water, which is consistent with the high water to air partition coefficient and its small, but detectable, presence in rain water, sea water, and lake water samples. Very little acetone is expected to reside in soil, biota, or suspended solids. This is entirely consistent with the physical and chemical properties of acetone and with measurements showing a low propensity for soil absorption and a high preference for moving through the soil and into the ground water.

In air, acetone is lost by photolysis and reaction with photochemically produced hydroxyl radicals; the estimated half-life of these combined processes is about 22 days. The relatively long half-life allows acetone to be transported long distances from its emission source.

Acetone is highly soluble and slightly persistent in water, with a half-life of about 20 hours; it is minimally toxic to aquatic life.

Acetone released to soil volatilises although some may leach into the ground where it rapidly biodegrades.

Acetone does not concentrate in the food chain.

Acetone meets the OECD definition of readily biodegradable which requires that the biological oxygen demand (BOD) is at least 70% of the theoretical oxygen demand (THOD) within the 28-day test period.

Drinking Water Standard: none available.

Soil Guidelines: none available.

Air Quality Standards: none available.

Ecotoxicity:

Testing shows that acetone exhibits a low order of toxicity.

Fish LC50: brook trout 6070 mg/l; fathead minnow 15000 mg/l

Bird LC0 (5 day): Japanese quail, ring-neck pheasant 40,000 mg/l

Daphnia magna LC50 (48 h): 15800 mg/l; NOEC 8500 mg/l

Aquatic invertebrate 2100 - 16700 mg/l

Aquatic plant NOEC: 5400-7500 mg/l

Daphnia magna chronic NOEC 1660 mg/l

Acetone vapors were shown to be relatively toxic to two types insects and their eggs. The time to 50% lethality (LT50) was found to be 51.2 hr and 67.9 hr when the flour beetle (*Tribolium confusum*) and the flour moth (*Ephestia kuhniella*) were exposed to an airborne acetone concentration of 61.5 mg/m³. The LT50 values for the eggs were 30-50% lower than for the adult. The direct application of acetone liquid to the body of the insects or surface of the eggs did not, however, cause any mortality.

The ability of acetone to inhibit cell multiplication has been examined in a wide variety of microorganisms. The results have generally indicated mild to minimal toxicity with NOECs greater than 1700 mg/L for exposures lasting from 6 hr to 4 days. Longer exposure periods of 7 to 8 days with bacteria produced mixed results; but overall the data indicate a low degree of toxicity for acetone. The only exception to these findings were the results obtained with the flagellated protozoa (*Entosiphon sulcatum*) which yielded a 3-day NOEC of 28 mg/L.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
acetone	LOW (Half-life = 14 days)	MEDIUM (Half-life = 116.25 days)
dimethyl carbonate	HIGH	HIGH
propane	LOW	LOW
iso-butane	HIGH	HIGH
propylene glycol monomethyl ether acetate, alpha-isomer	LOW	LOW
n-butanol	LOW (Half-life = 54 days)	LOW (Half-life = 3.65 days)
titanium dioxide	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
acetone	LOW (BCF = 0.69)
dimethyl carbonate	LOW (LogKOW = 0.2336)
propane	LOW (LogKOW = 2.36)
iso-butane	LOW (BCF = 1.97)
propylene glycol monomethyl ether acetate, alpha-isomer	LOW (LogKOW = 0.56)
n-butanol	LOW (BCF = 0.64)
titanium dioxide	LOW (BCF = 10)

Mobility in soil

Ingredient	Mobility
acetone	HIGH (KOC = 1.981)
dimethyl carbonate	LOW (KOC = 8.254)
propane	LOW (KOC = 23.74)
iso-butane	LOW (KOC = 35.04)
propylene glycol monomethyl ether acetate, alpha-isomer	HIGH (KOC = 1.838)
n-butanol	MEDIUM (KOC = 2.443)
titanium dioxide	LOW (KOC = 23.74)

SECTION 13 Disposal considerations**Waste treatment methods****Product / Packaging disposal**

DO NOT allow wash water from cleaning or process equipment to enter drains.

Continued...

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- It may be necessary to collect all wash water for treatment before disposal.
- In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
- Where in doubt contact the responsible authority.
- Consult State Land Waste Management Authority for disposal.
- Discharge contents of damaged aerosol cans at an approved site.
- Allow small quantities to evaporate.
- **DO NOT incinerate or puncture aerosol cans.**
- Bury residues and emptied aerosol cans at an approved site.

SECTION 14 Transport information**Labels Required****Land transport (ADG)**

UN number	1950	
UN proper shipping name	AEROSOLS	
Transport hazard class(es)	Class Subrisk	2.1 Not Applicable
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions Limited quantity	63 190 277 327 344 381 1000ml

Air transport (ICAO-IATA / DGR)

UN number	1950	
UN proper shipping name	Aerosols, flammable	
Transport hazard class(es)	ICAO/IATA Class ICAO / IATA Subrisk	2.1 Not Applicable
	ERG Code	10L
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions Cargo Only Packing Instructions Cargo Only Maximum Qty / Pack Passenger and Cargo Packing Instructions Passenger and Cargo Maximum Qty / Pack Passenger and Cargo Limited Quantity Packing Instructions Passenger and Cargo Limited Maximum Qty / Pack	A145 A167 A802 203 150 kg 203 75 kg Y203 30 kg G

Sea transport (IMDG-Code / GGVSee)

UN number	1950	
UN proper shipping name	AEROSOLS	
Transport hazard class(es)	IMDG Class IMDG Subrisk	2.1 Not Applicable
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	EMS Number Special provisions Limited Quantities	F-D , S-U 63 190 277 327 344 381 959 1000 ml

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
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Continued...

844AR Acrylic ESD Coating (Aerosol)

Product name	Group
acetone	Not Available
dimethyl carbonate	Not Available
propane	Not Available
iso-butane	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
n-butanol	Not Available
titanium dioxide	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
acetone	Not Available
dimethyl carbonate	Not Available
propane	Not Available
iso-butane	Not Available
propylene glycol monomethyl ether acetate, alpha-isomer	Not Available
n-butanol	Not Available
titanium dioxide	Not Available

SECTION 15 Regulatory information**Safety, health and environmental regulations / legislation specific for the substance or mixture****acetone is found on the following regulatory lists**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

dimethyl carbonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

propane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

iso-butane is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Chemical Footprint Project - Chemicals of High Concern List

propylene glycol monomethyl ether acetate, alpha-isomer is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

n-butanol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

titanium dioxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (acetone; dimethyl carbonate; propane; iso-butane; propylene glycol monomethyl ether acetate, alpha-isomer; n-butanol)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECL	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes

844AR Acrylic ESD Coating (Aerosol)

National Inventory	Status
Vietnam - NCI	Yes
Russia - FBEPH	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	26/01/2022
Initial Date	24/01/2022

SDS Version Summary

Version	Date of Update	Sections Updated
1.4	26/01/2022	Chronic Health, Classification, Fire Fighter (fire/explosion hazard), Physical Properties

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average
 PC – STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit.
 IDLH: Immediately Dangerous to Life or Health Concentrations
 ES: Exposure Standard
 OSF: Odour Safety Factor
 NOAEL: No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index
 AIIC: Australian Inventory of Industrial Chemicals
 DSL: Domestic Substances List
 NDSL: Non-Domestic Substances List
 IECSC: Inventory of Existing Chemical Substance in China
 EINECS: European INventory of Existing Commercial chemical Substances
 ELINCS: European List of Notified Chemical Substances
 NLP: No-Longer Polymers
 ENCS: Existing and New Chemical Substances Inventory
 KECL: Korea Existing Chemicals Inventory
 NZIoC: New Zealand Inventory of Chemicals
 PICCS: Philippine Inventory of Chemicals and Chemical Substances
 TSCA: Toxic Substances Control Act
 TCSI: Taiwan Chemical Substance Inventory
 INSQ: Inventario Nacional de Sustancias Químicas
 NCI: National Chemical Inventory
 FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances